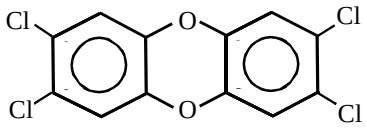
CHLORINATED DIOXINS AND DIBENZOFURANS

Identified as a toxic air contaminants under California's air toxic's program (AB 1807) in 1986.

CAS Registry Number for TCDD: 1746-01-6

Molecular Formula: $C_{12}H_4Cl_4O_2$



2,3,7,8 Tetrachlorodibenzo-p-dioxin

Dioxin is a generic term used to denote a single compound or mixture of compounds derived from polychlorinated dibenzodioxins. The basic structure of all dioxins consists of 2 benzene rings joined by 2 oxygen atoms. The most commonly known, and perhaps one of the most toxic compounds, is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (ARB, 1986a).

A closely related family of compounds are the polychlorinated dibenzofurans. They have structures similar to dioxins and are often found in association with them. The potency of dioxins and dibenzofurans is dependent on the chemical structure of the individual compound. The toxicity of various tetrachlorodioxins, for example, may vary by a factor of 100,000 or more. Dioxins and dibenzofurans chlorinated in the 2, 3, 7, and 8 positions have been demonstrated to be carcinogens (ARB, 1986a).

There are 75 different polychlorinated dibenzo-p-dioxins and 135 dibenzofurans which are classified into groups termed homologues on the basis of the number of chlorine atoms in the molecule. Chemical analysis is exceedingly difficult because environmental samples are composed of a large number of various chlorinated dioxins and dibenzofurans at very low concentrations (ARB, 1986a).

The mixture of dioxins and furans emitted from combustion sources are in both the gaseous and particulate phase. The persistence of these substances may be a function of the phase into which they are emitted. The gas/solid phase partition factor is influenced by flow rate, temperature, and dimensions of the sampling. These substances do not appear to degrade when sorbed to solids (ARB, 1986a). It is believed that the hexa through hepta chlorinated congeners are sorbed to particulates, whereas, the tetra and penta congeners partition to the vapor phase (Bidleman, 1988).

Physical Properties of 2,3,7,8-Tetrachlorodibenzo-p-dioxin

Synonyms: TCDD; dioxine; tetradioxin; 2,3,7,8-TCDD; tetrachlorodibenzodioxin

Molecular Weight:	322
Melting Point:	305 - 306 ^{o}C
Vapor Pressure:	7.4 x 10^{-10} mm Hg at 25^{o}C
Water Solubility:	19.3 ng/L at 22 ^{o}C
Thermal Decomposition:	700 ^{o}C
Log Octanol/Water Partition Coefficient:	7.02
Henry's Law Constant:	1.62 x 10^{-5} atm cu m/mole at 25^{o}C
Conversion Factor:	1 ppm = 13.17 mg/m^{3}

(HSDB, 1995; Marple et al., 1987; Merck, 1989; Sax, 1989)

SOURCES AND EMISSIONS

A. Sources

Discovery of dioxins in a variety of combustion processes has led some researchers to theorize that the production of dioxins is a general phenomenon associated with all combustion processes (Bumb et al., 1980). Dioxins are emitted from incinerators that burn medical waste, municipal solid waste, hazardous waste sewage sludge, tires, and metal smelting operations when the feedstock contains dioxin precursors (ARB, 1990e). Dioxins are formed in small quantities as unwanted combustion byproducts in certain industrial processes associated with the manufacture of polychlorinated biphenyls (PCBs), and as products of incomplete combustion when chlorine and complex mixtures containing carbon are present. Other possible sources of dioxins are sawmills, wire and scrap metal reclamation incinerators, black liquor boilers, cement kilns, cofiring wastes, transformer fires, wood stoves/fireplaces, and agricultural burning. Dioxin can form in wood through chlorination of phenolic compounds present in wood, paper pulp, or through the combustion breakdown of pentachlorophenol, a pesticide used to inhibit mold growth in lumber. 2,3,7,8-Tetrachlorodibenzo-p-dioxin is produced as an unwanted contaminant during the manufacture of pesticides, such as chlorophenols, and their derivatives such as 2,4,5-trichlorophenoxyacetic acid (ATSDR, 1987). Chlorinated dioxins and dibenzofurans have also been detected in fly ash and stack gas of various combustion processes (Tiernan, 1983).

Dioxin in very small concentrations is ubiquitous in the environment and it is likely that many of the primary sources are not yet known. Dioxins have been found worldwide, even in remote areas (ARB, 1986a).

Chlorinated dioxins and dibenzofurans adsorbed on airborne particulate or in industrial effluent are deposited on the soil and eventually bind to other organic substances and bottom sediment in lakes and rivers. Although dioxins are encountered in both the vapor and particulate phases, it has been suggested that ingestion results in 90 percent of human exposure (Gilman & Newhook, 1991). Atmospheric dioxins deposit on vegetation which farm

animals consume. Humans then ingest crops, fish, meat, and dairy products and thus accrue a body burden of dioxin. Subsistence fisherman can have unusually high levels of dioxin (U.S. EPA, 1989a; Hites, 1991). Secondary exposure, due to such soil and water pollution, may be as significant as atmospheric exposure and could substantially increase total risk (ARB, 1986a).

The primary stationary sources that have reported emissions of dioxins in California are sanitary services, manufacturers of medical instruments and supplies, and cement and hydraulics products. The primary stationary sources that have reported emissions of dibenzofurans in California are manufacturers of miscellaneous plastics products, sanitary services, and sawmills and planing mills (ARB, 1997b).

B. Emissions

The total emissions of dioxins and dibenzofurans from stationary sources in California are estimated to be at least 4.00 and 61 pounds per year, respectively, based on data reported under the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

In 1990, the Air Resources Board (ARB) adopted a control measure designed to reduce dioxin emissions from medical waste incinerators because they have the potential for the greatest individual risk of all dioxin sources. The regulation requires either a 99 percent reduction in dioxins emissions or achievement of an emission limit of 10 nanograms of dioxins per kilogram of waste burned. These reductions are to be achieved by: combustor design and operation, temperature control maintenance, use of operation logs, monitoring equipment, and source tests (ARB, 1990e). In 1994, the United States Environmental Protection Agency (U.S. EPA) adopted a control measure to regulate municipal waste incinerators by the year 2000 (U.S. Federal Register, 1994).

C. Natural Occurrence

Dioxins are not known to occur naturally in the environment (HSDB, 1995).

AMBIENT CONCENTRATIONS

When dioxin was formally identified as a toxic air contaminant in 1986, the ARB estimated a population-weighted annual concentration of 0.4 picograms per cubic meter (pg/m^3) for chlorinated dioxin and 1.0 pg/m^3 for chlorinated dibenzofurans. Because of limited data, these ambient concentrations were calculated on emissions estimates using air quality modeling for nine municipal waste incinerators proposed for construction in Southern California (ARB, 1986a).

The ARB commissioned a study to assess the ambient concentrations of polychlorinated dibenzodioxins and polychlorinated dibenzofurans in the South Coast Air Basin

(Hunt et al., 1990). 2,3,7,8-Tetrachlorodibenzo-p-dioxin levels were non measurable at some sites and 0.0086 pg/m^3 at West Long Beach (monitor near a petroleum refinery) and 0.034 pg/m^3 at the CalTrans site (monitor near a highway intersection) (U.S. EPA, 1993a).

A study to assess ambient concentrations of dioxins was also conducted in Fresno, California in 1991. The majority of the atmospheric burdens of PCDDs/PCDFs are represented by non 2,3,7,8-substituted species which are not of toxicological importance. However, the reported range for TCDD was 0.012 to 0.027 pg/m^3 and for 2,3,7,8-tetrachlorodibenzo-p-furans it was 0.041 to 0.134 pg/m^3. It is thought that combustion sources (including wood stoves as shown by high retene concentrations) are responsible for these emissions (ARB, 1993d).

The U.S. EPA has also reported a mean concentration of 0.04 pg/m^3 of dioxin from five study areas during 1986 to 1988 (U.S. EPA, 1993a).

INDOOR SOURCES AND CONCENTRATIONS

No information about dioxin indoor sources and concentrations was found in the readily available literature.

ATMOSPHERIC PERSISTENCE

Chlorinated dioxins and dibenzofurans are highly persistent under normal environmental conditions, particularly when adsorbed on soils or other substrates. Long distance transport of these materials in the atmosphere has been documented. The half-life of 2,3,7,8-TCDD has been reported to be approximately 25 to 100 years in subsurface soil and 9 to 15 years at the soil surface (Paustenbach et al., 1992). Dioxins are degraded by sunlight in solution under laboratory conditions, but the extent to which dioxins are degraded by sunlight in the atmosphere is unknown (ARB, 1986a). Gas-phase dioxins may be degraded by reaction with hydroxyl (OH) radicals and direct photolysis. The average half-life for particles in the troposphere is about 3.5 to 10 days estimate that lifetime and half-life of dioxins in the troposphere are several days, whether particle-associated or gaseous (Atkinson, 1995; Balkanski et al., 1993). Particulate-associated dioxins are removed from air by wet and dry deposition (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics “Hot Spots” Program (AB 2588). Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans were calculated as total TCDD equivalents in the AB 2588 risk assessments. Of the risk assessments reviewed as of April 1996, TCDD equivalents were the major contributor to the overall cancer risk in 11 of the approximately 550 risk assessments reporting a total cancer risk equal to or greater than 1 in 1 million and contributed to the total cancer risk in 42 of the these risk assessments. 2,3,7,8-Tetrachlorodibenzo-p-dioxin equivalents also were the major contributors to the overall cancer risk in 1 of the approximately 130 risk

assessments reporting a total cancer risk equal to or greater than 10 in 1 million, and contributed to the total cancer risk in 9 of these risk assessments (OEHHA, 1996a).

For non-cancer health effects, TCDD equivalents contributed to the total hazard index in 6 of the approximately 89 risk assessments reporting a total chronic hazard index greater than 1, and presented an individual hazard index greater than 1 in 1 of these risk assessments (OEHHA, 1996b).

HEALTH EFFECTS

Probable routes of human exposure to TCDD are inhalation and ingestion. Skin exposure can occur through contact with contaminated soils. Mothers milk may expose a nursing baby to 4 to 12 percent of the estimated lifetime dose (WHO, 1988). Once dioxin enters the human body, a small amount is metabolized and eliminated, while the rest bioaccumulates in body fat. As fat is metabolized, stored dioxin is released and excreted primarily in feces. The body's concentration is dependent on the rates of ingestion, elimination, and storage capacity of dioxin. The approximate half-life of dioxin in humans was estimated to range from 6 to 10 years (Pirkle, 1989).

Non-Cancer: Acute exposure of humans to dioxins has caused chloracne, liver toxicity, skin rashes, nausea, vomiting, and muscular aches and pains (Olson, 1994). A severe weight loss in animals has been observed following acute exposure to dioxin as have hyperkeratosis, facial alopecia, inflammation of the eyelids, and loss of fingernails and eyelashes. The immune system appears to be very sensitive to dioxin toxicity. Thymic atrophy is a prominent finding in exposed animals and has been observed in all laboratory species examined. Other lymphoid tissues such as the spleen, lymph nodes, and bone marrow are also affected (ARB, 1986a). Symptoms of chronic exposure to dioxins include splenic and testicular atrophy, elevated gamma-glutamyl transpeptidase levels, elevated cholesterol levels, and abnormal neurological findings (Moses et al., 1984). Other effects may include risk of enzyme induction, diabetes, and endocrine changes (Safe, 1990; Sweeny et al., 1992; Egeland et al., 1994).

A chronic, non-cancer Reference Exposure Level (REL) of 3.5×10^{-6} $\mu g/m^3$ is listed for chlorinated dibenzo-p-dioxins (as 2,3,7,8-equivalents) and chlorinated dibenzofurans (as 2,3,7,8-equivalents) by the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program, Revised 1992 Risk Assessment Guidelines. The toxicological endpoints considered for chronic toxicity are the immune, gastrointestinal and reproductive systems (CAPCOA, 1993). The U.S. EPA has not established a Reference Concentration (RfC) or an oral Reference Dose (RfD) for TCDD.

Human studies on the adverse reproductive and developmental effects of dioxins have proven inconclusive. Animal studies have shown TCDD to be both teratogenic and fetotoxic. Reproductive and teratogenic effects observed in animals are cleft palate, kidney abnormalities, decreased fetal weight, and survival, hydrocephalus, open eye, edema, resorptions, petechiae, and

infertility (ARB, 1986a). The State of California, under Proposition 65, has determined that TCDD is a developmental toxicant (CCR, 1996).

Cancer: Human studies which have reported cancer increases are inconclusive because of inadequate data. There is adequate evidence to support a conclusion that TCDD is carcinogenic in rodents and should be considered a potential carcinogen to humans (ARB, 1986a). Ingestion studies in rodents have shown increases in tumors of the liver, lung, squamous cell, nasal turbinates, and hard palate (ARB, 1986a).

The U.S. EPA is currently preparing a final Dioxin and Related Compounds risk assessment document. A workshop draft report was released to the public August 1994, and a public comment period is now in progress. The International Agency for Research on Cancer classified TCDD as Group 1: Human carcinogen, based on sufficient evidence in humans (IARC, 1997).

The State of California has determined under Proposition 65 that TCDD is a carcinogen (CCR, 1996). The inhalation potency factor that has been used as a basis for regulatory action in California is 38 (microgram per cubic meter)$^{-1}$ (OEHHA, 1994). In other words, the potential excess cancer risk for a person exposed over a lifetime to 1 picogram per cubic meter of dioxin is estimated to be no greater than 38 in 1 million (CAPCOA, 1993). The oral potency factor that has been used as a basis for regulatory action in California for TCDD is 1.3×10^5 (milligram per kilogram per day)$^{-1}$ (OEHHA, 1994).

There are 210 polychlorinated dibenzodioxin and dibenzofuran isomers. The various isomers are not equally toxic nor are they considered equally potent as carcinogens. For the purpose of assessing cancer risk associated with these chemicals, the U.S. EPA adopted a procedure in 1989 which uses the concept of toxic equivalent factors (TEF) (See Table I). The isomer TCDD appears to be the most potent of these chemicals. In the TEF scheme, TCDD is assigned a TEF of 1.0. The cancer potency of all other isomers chlorinated in the 2,3,7, and 8 positions are related to TCDD and TCDF. When calculating toxic equivalents, it is necessary to know the proportion of total dioxins and furans that is made up of the 15 isomers chlorinated in the 2,3,7, and 8 positions. The TEFs are used as input to a dispersion model to calculate risks from ambient concentrations for specified receptors in California risk assessments prepared under AB 2588 (CAPCOA, 1993).

Table I - Toxicity Equivalency Factors (TEF)
for Chlorinated Dibenzodioxins and Chlorinated Dibenzofurans

Compound	TEF
Mono-, Di, and Tri - CDDs	0

2,3,7,8-TCDD	1
Other TCDDs	0
2,3,7,8-PeCDD	0.5
Other PeCDDs	0
2,3,7,8-HxCDD	0.1
Other HxCDDs	0
2,3,7,8-HpCDD	0.01
Other HpCDDs	0
OCDD	0.001
Mono-, Di-, and Tri - CDFs	0
2,3,7,8-TCDF	0.1
Other TCDFs	0
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
Other PeCDFs	0
2,3,7,8-HxCDF	0.1
Other HxCDFs	0
2,3,7,8-HpCDF	0.01
Other HpCDFs	0
OCDF	0.001

(U.S. EPA, 1989a)